Archives of Disease in Childhood, 1980, **55**, 163–167

Correspondence

A suggested child-health clinic form

Sir,

I read with interest the paper by Professor Illingworth (*Archives*, 1979, **54**, 626). The format is clear and concise, and the columns for 'normal', 'doubtful', 'abnormal', and 'why the finding may be abnormal' are practical and serve to emphasise the author's oft repeated warning of the fallacies of a figure for an overall score. The list of risk factors, similar to that used in an earlier record card designed by a subcommittee of the BPA and the (then) Society of Medical Officers of Health, has proved invaluable.

My main points of criticism are:

(1) The omission of tests for distant and near vision in infants from 6 months. Two of the important questions asked of developmental assessment are, 'Can he hear?' and 'Can he see?'. Child health doctors have become increasingly aware of the need to attempt to answer the former but still require guidance to learn to use Stycar tests of vision, admirably designed by Mary Sheridan. The visual experience of young children plays an important part in early learning. Poor visual acuity is not necessarily associated with nystagmus, opacity, or persistent squint and may be missed unless tests for vision are done routinely.

(2) The development of prelanguage and early language skills is of special importance from 9 months. By 2 years the child's personality is becoming more integrated and any unevenness between performance and language skills may be significant. A simple heading 'Speech' is inadequate in this context. The whole cognitive development of the child is closely bound up with the ability to communicate in spoken language. There are many reasons why a child may ultimately fail to develop expressive speech. The child health doctor must learn to think of language development in terms of comprehension, symbolisation, and expressive speech if effective intervention programmes are to be introduced early.

(3) The subheading 'Developmental examination only if necessary', from 2 years, is difficult to interpret and appears contrary to the aim for periodical developmental assessment of all young children.

There is little doubt that a well-designed record form can offer helpful guidance and contribute to a rise in the standard of the service itself. Professor Illingworth has offered a design which goes some way towards this objective.

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Professor Illingworth comments:

I am pleased to have Dr Egan's observations. The omission of tests for distant and near vision in infants was deliberate. I should like to know what is to be gained by the tests recommended by Dr Egan when there is no squint, opacity, or nystagmus, and when it is clear from watching the child reach out and grasp small objects that there is no significant treatable visual defect.

Regarding the heading 'Speech', I should like to see a clear statement of the 'effective intervention programmes' advised by Dr Egan for a child, starting to speak later than usual and who has been properly tested for a hearing defect, with the evidence for the effectiveness of that programme.

I agree that the words 'developmental examination only if necessary' should be changed.

I should emphasise that in a busy clinic (like mine where, single-handed, I see at least 40 babies in an afternoon session) one must be sensible and perform only the tests which are useful and important. If the routine rapid screening reveals some doubt about normality, detailed examination of visual or other functions is essential and then one may have to devote an hour or more to a single child.

Dr Irene Chesham has pointed out a significant omission on the suggested chart; a test for phenylketonuria should have been included.

The form was intended to be for consideration, and I shall be grateful for all constructive criticism.

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Wilson's disease, chronic copper poisoning, or Indian childhood cirrhosis?

Sir,

In 1973, the case was described of an Australian boy who presented at age 14 months with abdominal distension.[1] He was found to have a micronodular cirrhosis, renal tubular aminoaciduria, and anaemia. He became jaundiced and died 6 weeks later. Histological examination of the liver showed continuing parenchymal destruction, no regeneration, cytoplasmic hyaline changes, and copper storage. The necropsy hepatic copper concentration exceeded 3000 μg/g dry weight; 47 μmol/g (normal range 15–55 μg/g; 0·24–0·87 μmol/g).

Had this been a Hindu child living in India, his clinical features would have been regarded as typical of Indian childhood cirrhosis (ICC), in which condition hepatic

copper is also grossly raised.[2] We were able to re-examine the necropsy histology of this child, and found it indistinguishable from the changes seen in ICC.[3]

The boy's copper toxicosis was attributed to the ingestion of water which had a high copper concentration. Although the family's well water contained little copper, it had a pH of 3·8–4·8, and dissolved copper from the domestic plumbing. Three explanations may be offered for the fact that his parents and his 4-year-old brother were unaffected. Firstly, he may also have had Wilson's disease, as suggested by a plasma caeruloplasmin concentration of 150 mg/l, although this may not be significant in the presence of a serum albumin concentration of 21 g/l. Plasma caeruloplasmin concentration is normal in patients with ICC.[4] Secondly, since the neonate has a modestly increased hepatic copper content,[5] there may be greater susceptibility to copper toxicosis in infancy. Thirdly, he had been fed on cows' milk diluted with water, and it is known that the availability for absorption of copper in milk is greater than in other foods.[6]

This case report therefore shows that a disease state strikingly similar to ICC may be produced by excessive copper ingestion, but leaves unresolved the question—Can this occur in normal infants or only in those genetically predisposed?

We thank Dr Pat Bale, Royal Alexandra Hospital for Children, Camperdown, New South Wales, for providing the necropsy specimens.

References

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Pneumopericardium in a neonate not artificially ventilated

Sir,

Because the incidence of pneumopericardium in neonates has been increasingly reported since the advent of positive pressure-assisted ventilation in the treatment of hyaline membrane disease, we report a case of pneumopericardium in a neonate in the absence of any form of assisted ventilation.

A 1766-g baby boy was the first of twins born to a 24-year-old gravida 3, para 2 female whose pregnancy was complicated by spotting and a urinary tract infection in the first trimester. The infant was delivered vaginally without difficulty. The Apgar scores were 7 at one minute and 7 at five minutes. On admission to a nursery at another hospital, the infant appeared to be cyanotic and plethoric and had grunting respirations. He was given oxygen by hood to relieve cyanosis. Haemocrit was 80%, assumed to be high on the basis of feto-fetal transfusion. He was phlebotomised 15 ml and this volume was replaced with Plasmanate (Cutter Laboratories) resulting in a haemocrit of 69%. He was then transferred to the Children's Mercy Hospital where the initial examination showed a plethoric infant of about 35 weeks' gestation. He was in mild respiratory distress with audible grunting and intercostal retraction. The initial chest *x*-ray at age 8 hours showed a fine reticulogranular pattern in both lungs with slight diffuse atelectasis (Fig. 1). Arterial blood-gases and pH on 40% oxygen by hood were pH 7·28, P_{CO_2} 48 mmHg (6·4 kPa), and P_{O_2} 90 mmHg

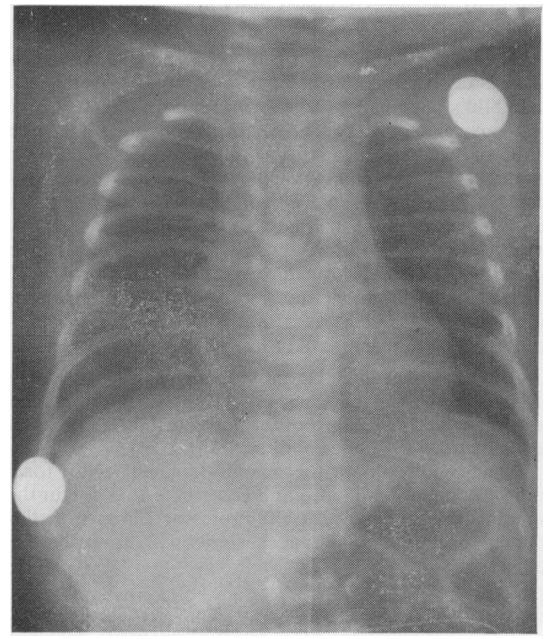

Fig. 1 *Chest x-ray at 8 hours showing fine reticulogranular pattern of diffuse atelectasis.*